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Constraint Logic Programming; 4.8 Beyond Finite Domains; 4.9 Distributed Constraint Programming; 4.10 Application Areas; 4.11 Conclusions; Bibliography; Chapter 5. Conceptual Graphs; 5.1 From Existential Graphs to Conceptual Graphs; 5.2 Common Logic; 5.3 Reasoning with Graphs; 5.4 Propositions, Situations, and Metalanguage; 5.5 Research Extensions; Bibliography; Chapter 6. Nonmonotonic Reasoning; 6.1 Introduction; 6.2 Default Logic; 6.3 Autoepistemic Logic; 6.4 Circumscription; 6.5 Nonmonotonic Inference Relations 6.6 Further Issues and ConclusionAcknowledgements; Bibliography; Chapter 7. Answer Sets; 7.1 Introduction; 7.2 Syntax and Semantics of Answer Set Prolog; 7.3 Properties of Logic Programs; 7.4 A Simple Knowledge Base; 7.5 Reasoning in Dynamic Domains; 7.6 Extensions of Answer Set Prolog; 7.7 Conclusion; Acknowledgements; Bibliography; Chapter 8. Belief Revision; 8.1 Introduction; 8.2 Preliminaries; 8.3 The AGM Paradigm; 8.4 Belief Base Change; 8.5 Multiple Belief Change; 8.6 Iterated Revision; 8.7 Non-Prioritized Revision; 8.8 Belief Update; 8.9 Conclusion; Acknowledgements; Bibliography
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Chapter 11. Bayesian Networks

Sommario/riassunto

Knowledge Representation, which lies at the core of Artificial Intelligence, is concerned with encoding knowledge on computers to enable systems to reason automatically. The Handbook of Knowledge Representation is an up-to-date review of twenty-five key topics in knowledge representation, written by the leaders of each field.This book is an essential resource for students, researchers and practitioners in all areas of Artificial Intelligence.* Make your computer smarter* Handle qualitative and uncertain information* Improve computational tractability to solve yo

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Autore	Gilles J. Guillemin
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Sommario/riassunto

Immune responses within the brain are still scarcely explored. Nerve tissue damage is accompanied by the activation of glial cells, primarily microglia and astroglia, and such activation is responsible for the release of cytokines and chemokines that maintain the local inflammatory response and actively recruit lymphocytes and monocytes to the damaged areas. Theoretically, these responses are designed to repair the brain damage. However, alterations, or a chronic perpetuation of these responses may underlie a number of neuro-pathologies. It is thought that each inflammatory scenario within the brain have a specific biochemical footprint characterized by the release of determined cytokines, chemokines and growing factors able to define particular immunological responses. Alongside, glial cells transform their cell body, become larger and develop higher number of branches adopting an active morphological phenotype. These changes are related with the search of interactions with other cells, such as bystander resident cells of the brain parenchyma, but also cells homing from the blood stream. In this process, microglia and astrocytes communicates with other cells by the formation of specific intercellular connections that are still poorly understood. These interactions are complex and entail the arrangement of cytoskeletal compounds, secretory and phagocytic domains. In this particular crosstalk there is a two-way communication in which glial cells and target cells come together establishing interfaces with specific information exchange.

This way, glial cells orchestrate the particular response recruiting cellular subsets within the central nervous system and organizing the resolution of the brain damage. In this Frontiers Research Topic, we compile a selection of articles unfolding diverse aspects of glial-derived inflammation, focused on neurodegenerative diseases and other nervous system disorders, with special emphasis on microglia/macrophages as leading actors managing neuro-immunity.